MECHANICAL FRAGMENTATION OF PULMONARY THROMBOEMBOLI IN DOGS BY MEANS OF A FLEXIBLE ROTATING TIP CATHETER (KENSEY CATHETER).

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Pulmonary thromboembolism was induced in 10 dogs by the injection of 3-4 day old autologous blood clots. The clots were made radiopaque by soaking them 1 or 2 days in contrast material. The resulting clots were firm, 3-4 cm long, and 1 cm in diameter. Injection of the clots into the external jugular vein consistently produced occlusion of an intermediate or lobar pulmonary artery. The clots being radiopaque were readily visualized by image intensification fluoroscopy. Cine angiograms (35 mm cine, 30 frames/sec.) were taken before and after pulmonary embolism. In every incidence in which the Kensey catheter could be positioned at the site of obstruction, the clot was readily fragmented with a number 8 French (2.67 mm O.D.) Kensey catheter activated at 80,000 rpm. Activation time was usually 10-20 sec. Overall perfusion was shown by post-treatment angiograms to be markedly improved. There were no perforations of the pulmonary artery. In 5 dogs, we could not reach the clot. These studies show that catheter-tip fragmentation of pulmonary embolism with a Kensey catheter has excellent potential for emergency treatment. Second generation catheters will be fabricated with a fixed curve and with sufficient torque to improve the ability to quickly and easily position the catheter in branch arteries.

MARKED REDUCTION OF PROCOAGULANT ACUTE PHASE PROTEINS AFTER EARLY CORONARY THROMBOLYSIS

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The plasma kinetics of two procoagulant acute-phase proteins, plasminogen activator inhibitor-1 (PAI) and von Willebrand factor (vWF), were measured after successful or failed coronary thrombolysis. In 24 patients with acute myocardial infarction (AMI) of less than 6h duration, coronary patency was assessed angiographically before and 90 min after the start of intravenous tissue-plasminogen activator (t-PA). Continuous ECG recordings for ST segment changes and 4-hourly plasma MB-creatine kinase (CK-MB, $IU.l^{-1}$.24h) were obtained over the first 24h. PAI activity (parabolic-rate chromogenic assay, $IU.ml^{-1}$), vWF (ELISA, % normal plasma) and plasma C-reactive protein (CRP, $mg.l^{-1}$) were measured on admission (0h) and daily up to 72h. At 90 min from the start of t-PA the infarct-related artery was patent in 16 patients and occluded in 8. PAI and vWF (mean±SEM) in the 2 groups were :

	PAI				vWF			
	0h	24h	48h	72h	0h	24h	48h	72h
Patent	15±3	31±7	10±2	7±1	143±16	124±9	141±16	110±10
Occluded	13±4	38±2	25±11	51±25	157±18	206±23	190±25	201±41
p (t test)	NS	NS	<0.05	<0.02	NS	<0.001	NS	<0.01

In the patent group, time to reach one half of maximal ST elevation from the start of t-PA was 1.4±0.4h, integrated 24h CK-MB was 2106±373 and peak CRP 39±6; in the occluded group these were 7±3.7h ($p<0.04$), 4295±1364 ($p<0.05$) and 87±25 ($p<0.01$), respectively.

These results suggest that the acute-phase response of PAI and vWF to AMI is markedly reduced after early coronary thrombolysis. The curtailed response appears related to a shorter duration of ischemia and smaller infarct size. Prompt and successful coronary thrombolysis, by attenuating the early postinfarction procoagulant state, may decrease the incidence of thromboembolic complications soon after AMI and contribute to the maintenance of coronary patency.

IN VITRO EFFECTS OF THROMBOLYTIC AGENTS ON PLATELETS

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Whether the reported in vivo platelet activation during thrombolysis with streptokinase (SK) and TPA is due to a direct effect of the thrombolytic agents on platelets or to increased platelet-vessel wall interaction is unclear. We therefore assessed the direct effects of SK, TPA and urokinase (UK) on platelets in vitro. With both ADP (1 µmol/l) and collagen (1 µg/ml) as aggregating agents, SK at ≥100 units/ml led to a significant ($p<0.05$) mean reduction in the rate of platelet aggregation (PA). With collagen and, in most instances with ADP, this reduction in the rate of PA was associated with a decreased extent of PA. In 5 out of 30 cases, however, with ADP as aggregating agent, a conversion from reversible to irreversible PA occurred with SK. Thus, PA seemed to be stimulated by SK in these cases. TPA uniformly inhibited PA at ≥ 1 µg/ml with ADP and at ≥ 3.3 µg/ml with collagen as aggregating agent. UK inhibited PA at ≥300 units/ml with both aggregating agents. Platelet inhibition by the thrombolytics still occurred when the platelets were suspended in saline and, thus, does not seem to be dependent on plasmatic factors. Platelet synthesis of both thromboxane after stimulation with collagen, and c-AMP after prostaglandin E_1 was markedly reduced by either agent.
Thus, SK, TPA and UK inhibited platelets in vitro, although with SK, in a minority of cases, platelet stimulation was observed. In vivo platelet activation during thrombolysis should therefore not be due to a direct effect of the thrombolytic drugs on platelets.

PREVALENCE AND EMBOLIC POTENTIAL OF LEFT VENTRICULAR THROMBI IN DILATED CARDIOMYOPATHY.

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Thrombi may form in dilated cardiomyopathy (DCM) resulting in embolization, but the prevalence is unknown. To determine the prevalence and embolic potential of LV thrombi in DCM we prospectively studied 25 unselected patients with dilated, globally hypokinetic, ventricles unrelated to coronary disease who were not receiving anticoagulation. 115 2-dimensional echocardiograms were performed 3-monthly for a mean of 22 months. LV thrombi were present on ≥ 1 echocardiogram in 14 (56%) patients, were generally small and sessile and tended to persist when present. Fractional shortening (FS) was ≤ 10% in 15 patients of whom 12 (80%) had an LV thrombus compared to 3/10 (30%) with FS 11-24% ($p<0.05$). Five patients had a clinical embolus (stroke 4, coronary embolus 1) of whom 4 had a previously visualized thrombus which protruded in 3. NYHA class did not predict embolus. Warfarin anticoagulation was instituted in 4/5 with no further events at a mean anticoagulated follow-up of 15 months. CONCLUSIONS: 1) In dilated cardiomyopathy LV thrombi are usually small and thus may be overlooked because of adjacent trabeculae. 2) Thrombus in DCM is most common when FS is ≤ 10%, but symptoms do not predict risk of thrombus or embolus. 3) Protrusion although uncommon, may be a marker for embolus 4) Prophylactic anticoagulation to prevent emboli in DCM appears logical but trials are necessary to assess the risk-benefit ratio, since bleeding risks may be increased by associated hepatic congestion.